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IN THE UNITED STATES PATENT AND TRADEMARK OFFICE

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TECH CENTER 1600/2900

In Application of: Kaser et al.

Title: POLYCYCLIC AROMATIC HYDROCARBON INDUCED MOLECULES

Serial No.: 09/838,044

Filing Date: April 18, 2001

Examiner: Liu, S

Group Art Unit: 1653

Box Non-Fee Amendment

Commissioner for Patents

Washington, D.C. 20231

TRANSMITTAL FEE SHEET

Sir:

Transmitted herewith are the following for the above-identified application:

1. Return Receipt Postcard;
2. Response to Office Action (19 pp.);
3. Declaration under 37 CFR 1.131 (5 pp.);
4. Exhibits A-E;
5. References 1, 4, 5 and 9; and
6. Kikuno et al. reference.

The fee has been calculated as follows:

Claims	Claims After Amendment	-	Claims Previously Paid For	=	Present Extra	Other Than Small Entity Rate	Fee	Additional Fee(s)
Total	5	-	20	=	0	x\$18.00	0	\$ 0
Indept.	1	-	3	=	0	x\$84.00	0	\$ 0
First Presentation of Multiple Dependent Claims:						+280.00	0	\$ 0
Total Fee:								\$ 0

☒ Please charge Deposit Account No. **09-0108** in the amount of :

\$ _____

The Commissioner is hereby authorized to charge any additional fees required under 37 CFR 1.16 and 1.17, or credit overpayment to Deposit Account No. 09-0108. **A duplicate copy of this sheet is enclosed.**

Respectfully submitted,

INCYTE GENOMICS, INC.

Date: November 6, 2002

David G. Streeter, Ph.D.

Reg. No. 43,168

Direct Dial Telephone: (650) 845-5741

Customer No.: 27904

3160 Porter Drive

Palo Alto, California 94304

Phone: (650) 855-0555

Fax: (650) 845-4166



Docket No.: PB-0011-1 DIV

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By: *Margaret M. Gasson* Printed: Margaret M. Gasson

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RESPONSE TO OFFICE ACTION

Sir:

This paper is responsive to the Office Action dated August 13, 2002, Applicant(s) request reconsideration of the above-referenced patent application in view of the following amendments and remarks.

IN THE SPECIFICATION

Please replace the paragraph beginning at page 2, line 17, with the following rewritten paragraph:

B1
There is growing evidence that predisposition to cancer may reside in polymorphic genes involved in carcinogen metabolism and repair. One major goal of epidemiologists is the identification of individuals who are exposed to high levels of carcinogen, carry cancer-predisposing genes, and lack protective factors. A combination of cancer-predisposing genes could be used as an intermediate risk marker rather than taking diagnosis of cancer as the endpoint. Such markers may include PAH-DNA adduct level and polymorphism in PAH-metabolizing enzymes such as the cytochrome P450 family member, cytochrome P4501A1, (CYP1A1), the 4 S PAH-binding protein glutathione S-transferase (GSTM1), and cAMP-